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BENEFIT OF ATORVASTATIN RELOAD ON ENDOTHELIAL PROGENITOR CELLS IN PATIENTS ON CHRONIC STATIN TREATMENT UNDERGOING PCI

Oral Contributions

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Background: In previous RCTs, atorvastatin pretreatment reduced periprocedural myocardial infarction in patients (pts) undergoing percutaneous coronary intervention (PCI). Statins may promote mobilization of endothelial progenitor cells (EPCs), which may contribute to myocardial protection. Aim of this study was to investigate whether atorvastatin reload increases mobilization and function of EPCs in pts on chronic statin therapy undergoing PCI.

Methods: The study included two phases: A and B. In phase A, 53 pts on chronic statin therapy were randomized to receive an atorvastatin reload (80 mg 12 h before PCI, followed by 40 mg 2 h before PCI, N=27) or placebo (N=26). EPCs (CD45dim/ CD34+/CD133+/KDR+ cells) level was determined by flow cytometry, according to the ISHAGE protocol, on blood samples drawn at randomization, immediately before PCI, 8 and 24 h later. In phase B, 50 pts were randomized to receive atorvastatin reload with same modality of phase A (N=25) or placebo (N=25). We evaluated EPCs function by Hill Colony Forming Unit (CFU) Assay, which was expressed as EPC CFU/well. Blood samples for CFU Assay were drawn at randomization and 24 h after PCI. Primary endpoint was changes in EPCs levels and EPC CFU number/well after PCI according group of treatment.

Results: In phase A, EPCs levels were not different at baseline ($0.32 \pm 0.25\%$ of total events in reload vs $0.26 \pm 0.24\%$ of total events in placebo arm, $P=0.33$). Before PCI, EPCs level was significantly higher in the reload arm ($0.76 \pm 1.04\%$ vs $0.25 \pm 0.29\%$; $P=0.02$). Significantly higher EPCs levels were also observed in the reload arm at 8 h ($0.88 \pm 1.37\%$ vs $0.25 \pm 0.20\%$; $P=0.02$) and 24 h ($0.80 \pm 1.01\%$ vs $0.25 \pm 0.23\%$; $P=0.009$). In phase B, number of EPC CFU/well at baseline was not significantly different (9.4 ± 6.6 EPC CFU/well in reload arm vs 13.7 ± 9.2 EPC CFU/well in placebo arm, $P=0.07$). However, 24 h after PCI, number of EPC CFU/well was significantly higher in the reload arm (16.4 ± 8.6 vs 8.7 ± 8.2 EPC CFU/well, $P=0.002$).

Conclusions: High-dose atorvastatin reload prior to PCI promptly and significantly increases mobilization and function of EPCs. This may provide new insights into the mechanisms of cardioprotection afforded by statins in the setting of PCI.